Fluoridation: Do We Want It?

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SOME BAD EFFECTS OF FLUORIDATED WATER

Reported symptoms of fluoride poisoning are condensed below from Exner (6) and Waldbott (32):

1. Dental fluorosis, ectopic calcific deposits in pulp, periodontal disease.

2. Osteosclerosis, osteomalacia, exostoses, abnormal calcium deposits in and around joints and ligaments; together with severe nerve and muscle damage, which often produce disabling effects—including "poker-spine", which, however, may take as much as 40 years to develop. X-ray changes in poker back are severe.

3. Pain and stiffness in joints without x-ray changes.

4. Muscle pains, tenderness, weakness, shortness of breath, painful breathing.

5. Incoordination, altered reflexes, paresthesias, hearing loss or diminution.

6. Nausea, vomiting, diarrhea, anorexia.

7. Sterility, abortions, miscarriages, stillbirths.

8. Stunted growth, bow-legs.

9. Anemia, prolonged bleeding time, abnormal WBC counts and sedimentation rates.

10. Impaired liver and kidney function, goiter, cachexia, inanition and premature aging.

11. Taylor's work at the University of Texas (76) shows shortening of life spans.

Lemmon (77), pediatrician, of Amarillo (3.9 ppm F), Texas, reported: "Some of the babies have more tendency to bowing of legs, even in the face of constant anti-rachitic therapy, thus supporting the theory that toxic fluorides interfere with bone and dental metabolism."

KNOWN CASES OF FLUORIDE POISONING

Waldbott (32) described 52 cases of chronic fluoride poisoning from drinking water at or about 1 ppm fluoride.

Linsman & McMurray (78) gave an account of a death of an army sergeant from osteosclerosis due to drinking water. His first 7 years were spent in Spur (1.2 ppm), Texas, where he developed **severely mottled** teeth. The next two years were in Post (5.7 ppm), Texas, and then he lived for 7 years in Lubbock, Texas (4.4, ppm), moving to Washington (then unfluoridated), D. C., at 16 years of age. At 18 he returned to Lubbock, where he entered the army. He died of fluoride osteosclerosis at 22 years. A proper diagnosis would never have been made if an extraordinary post-mortem examination had not been done.

Pandit, et al (79), report the same diseased condition as Shortt (64), finding some cases occurring at fluoride concentrations as low as 0.6 ppm. Affected children, apart from mottling, showed no signs of fluoride poisoning. At about 25-30 years a gradual onset of symptoms began: tingling, pain and stiffness over the body, resulting limitation of mobility of spine and thorax. At 30-40 years of age, the final stages appeared: cachexia, emaciation, pressure symptoms on spinal cord, impotence, loss of sphincter control, mental impairment. Finally patients are bed-ridden with death coming from intercurrent disease. Important to note is that children slated for toxic manifestation show no signs of what is to come, except mottling.

FLUORIDE HARD ON HEART

Greenwood, et al (27), at Logan, Utah, studying effects of airborne fluorides, found fluoride deposits in the aorta. They noted: "As the degree of calcification of the aorta increased, there was a corresponding increase in the fluoride level. Fluorides are deposited along with calcium and phosphorus . . . the fluoride content of the aorta was higher than that of other soft tissues."

Ebert (80), studying metabolic pathways by which organs form embryologically, used sodium fluoride as an enzyme inhibitor. In low concentrations, it blocked, almost completely, the regions destined to form muscle, primarily affecting developing **heart** muscle. In high concentrations, it caused the entire embryo to disintegrate in a clear-cut pattern, starting with the heart-forming region. This causes us to remember that in the first 3 years of fluoridation, New Britain's (52) stillbirth rate jumped 150% while nearby unfluoridated Waterbury's rate remained unchanged.

Okushi (81) and Kono, et al (82), Japanese medical research men, produced severe myocardial damage in rats and white rabbits by feeding them 5-50 ppm F in foods. While these concentrations are greater than those recommended for fluoridation, the point is that in low concentrations, even less than 1 ppm, fluoride is cumulative and there may be damage as a result, damage that only controlled "in vivo" experiments will reveal.

FLUORIDE HARD ON KIDNEYS

Ramsyer (8) examined, at 520 days (equivalent to 42 years human age), 86 rats killed after living on drinking water at 1 ppm F continuously from birth. All of these animals had periodontal and questionable kidney lesions and all had more tooth decay than the controls. He concluded that fluorides at 1 ppm do not prevent tooth decay but that they do cause kidney and periodontal damage.

Rapaport (90), investigating the incidence of monogoloid births in Wisconsin, Illinois, and the Dakotas, found a definite relationship between the concentration of fluorine in the drinking waters and the frequency of mongolism, As fluoride concentrations were higher, incidence of **mongoloid births rose proportionately** and, further, with rising fluoride levels, the age of mothers producing mongoloid children declined. **These observations need no further comment.**

Considering the mass of published evidence against fluoridation, it is strange to find astute medical men, like the composers of the St. Louis Medical Society Report, announcing that "There is no published record of any injury to the health of any person . . . etc." It is disturbing to see continued insistence on the utilizing of mass treatment of populations through water supply fluoridation, when **such mass treatment is neither necessary nor advisable. It is also disquieting to hear capable scientists called "incompetent" and "biased," merely because their findings and beliefs happen to conflict with proponent scientists.** There unfortunately appears to be no climate of professioual opinion relative to fluoridation. **Scientific findings and opinions cannot be dismissed by discrediting and personally directed slander.**

CONSIDERATION OF "EVIDENCE" THAT FLUORIDATION WILL REDUCE DECAY BY 65-70%.

As proof of the efficacy of fluorides in decay reduction, proponents point to the "controlled" experiments at: Newburg-Kingston, Grand Rapids-Muskegon, Evanston-Oak Park (in the U.S.A.) and Brantford-Sarnia (in Canada).

In **scientifically** operated experiments, caluclated to determine the possible value of fluoridation, Sutton declares (83) the following basic conditions ought to be as nearly as possible satisfied:

1. Selected test (fluoride) and control (non-fluoride) cities must exhibit similarity in: size, climate, chemical composition of water supply, socio-economic status, basic (pre-test) decay rates and fluctuations therein.

2. Basic decay rates and fluctuations therein must be determined, for **both** test and control cities, **prior** to commencement of fluoridation in either.

3. A randomization procedure should be used to determine which city shall be test and which the control.

4. Decay rates should be taken in **both** test and control cities at regular times all throughout the test periods.

5. Groups of children for comparison should be of similar age, number, racial derivation —so as to prevent weighting of results (DMF rates).

6. The same examiners should conduct all examinations, examiners should be dentists, and they should use a standardized examination procedure.

7. Examiner bias should be eliminated by having examiners unaware of whether the children they are examining come from test or control areas.

8. Continuous residency for all children, test and control, should be ascertained.

9. Statistical adjustment procedures and devices should be standardized in all cases.

By these standards, our experiments are fiascos. In no case were basic decay rates determined for **both** cities before commencement of fluoridation in the test city. This meant that experimenters **did not** know whether the paired cities had similar basic decay rates until **after** experiments had commenced.

Fluctuations in basic decay rates were established prior to commencement of experiments in no instance. A randomization procedure was in no case utilized.

Water analyses were stated in no case beyond fluoride level determination. It was stated, in the Newburgh-Kingston case, that the waters for the two cities were similar, but here is how similar the 1952 U.S. Geological Survey shows them to be:

Newburgh (test), 35.0 ppm (calcium), 3.6 ppm (magnesium), 102.0 ppm (total hardness.

Kingston (control), 6.6 ppm (calcium), 0.9 ppm (magnesium), 20.0 ppm (total hardness).

Dissimilarities are here obvious. Newburgh fares much better in both calcium and magnesium, minerals essential for good tooth and bone formation.

THE SUNDAY ENTERPRISE (Beaumont, Texas)